CASE REPORT

A 21-year-old woman with mediastinal mass and cardiac arrest

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SUMMARY

Mediastinal mass syndrome (MMS) is a devastating respiratory and haemodynamic condition that might be encountered postintubation if special precautions are not carefully undertaken. We describe a case of MMS in a 21-year-old woman with a fatal outcome following emergency intubation for acute respiratory failure.

BACKGROUND

Mediastinal mass syndrome (MMS) has a variable spectrum of presentations. It has been associated with perioperative respiratory and circulatory life-threatening complications after induction of general anaesthesia and initiation of positive pressure ventilation. These complications mostly arise owing to the compression effect of the mass on the main bronchi and pulmonary arteries. A suitable approach for this kind of case is discussed in our report.

CASE PRESENTATION

A 21-year-old woman presented to the emergency department of our facility with dry cough and shortness of breath on minimal exertion. Her dyspnoea was worse in the supine position. Four months previously, the patient had been diagnosed with anterior mediastinal mass. Ultrasound guided biopsy showed B-cell malignant lymphoma. There was no history of fever, chest pain, cyanosis or wheezing.

On presentation, the patient was afebrile, with blood pressure of 117/81 mm Hg and a regular pulse of 140 bpm. The respiratory rate was 28/min with oxygen saturation of 95% on room air. Heart sounds were diminished, while lung auscultation showed a reduced right side air entry. The rest of the physical examination was normal.

INVESTIGATIONS

The chest radiograph revealed an enlarged mediastinum and a significant right-sided pleural effusion (figure 1). The white cell count was $15 \times 10^9/L$ with neutrophils and a normal haemoglobin level. Arterial blood gas on room air showed pH of 7.45, partial pressure of carbon dioxide of 30 mm Hg, partial pressure of oxygen of 60 mm Hg and oxygen saturation of 95%. Troponin T and pro-brain natriuretic peptide were normal. ECG showed sinus tachycardia.

A CT scan of the chest showed a large anterior mediastinal mass encasing the great vessels, including the right upper pulmonary artery and superior vena cava. There was at least 50% tracheal compression by mass. Furthermore, the right lung was

collapsed, with massive pleural effusion and pericardial effusion (figure 2).

The patient was admitted to the cardiothoracic ward. Oxygen supplement and maintenance intravenous fluids were provided. Within the first 24 h of presentation, the patient continued to be tachycardic and tachypnic, with no further deterioration. A right-sided ultrasound guided thoracentesis was performed, and a size 6 French catheter was placed inside the pleural cavity for continuous drainage. Only 1.5 litres of yellow blood-tinged pleural fluid was drained over 3 h. Subsequently, the patient developed a severe respiratory distress and productive cough with frothy secretions. Oxygen saturation decreased to 85–90% on high-flow oxygen supply. The blood pressure was 100/50, heart rate was 140 bpm and respiratory rate was 35 breaths/min. The patient was then transferred to the intensive care unit (ICU). No blood gases were obtained at this point as the condition of the patient declined quickly. As the patient arrived at the ICU, there was a rapid decline in her status that prevented the team from obtaining blood gases in a timely manner.

DIFFERENTIAL DIAGNOSIS

- Mediastinal mass syndrome
- Cardiac tamponade

TREATMENT

In the ICU, a decision was made to intubate her after a 30-min trial of non-invasive positive pressure ventilation, which did not show any clinical improvement.

Figure 1 Chest roentgenogram showing an enlarged mediastinum with bilateral pleural effusion.

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The patient was preoxygenated and premedicated with intravenous fentanyl 100 µg and ketamine 100 mg. Intravenous 50 mg of propofol was injected to achieve adequate sedation.

Figure 2 Chest CT scan with intravenous contrast showing a huge heterogeneous anterior mediastinal mass with at least 50% compression of the trachea. There is significant compression on the superior vena cava, subclavian veins, main pulmonary artery and its right branch. Massive right side pleural effusion and mild left side pleural effusion. Collapse of the right lung and small collapse of the left lower lobe. Moderate pericardial effusion.

The patient was placed in a supine position, and a 7.5 mm sized endotracheal tube (ETT) was easily placed through the vocal cords under direct visualisation by a laryngoscope. The tube position was confirmed by a capnogram and auscultation. Manual ventilation was conducted with little difficulty, using a self-inflating bag. Nonetheless, the patient continued to deteriorate with severe cyanosis, increased ETT secretions and sinus tachycardia. Intravenous 50 mg of atracurium was given to facilitate ventilation. Oxygen saturation declined to as low as 70–80%, and the patient developed severe hypotension. Intravenous 200 mg of phenylephrine was given. Shortly afterwards, the patient developed sudden cardiopulmonary arrest with pulseless electrical activity. Cardiopulmonary resuscitation (CPR), according to advanced cardiac life support guidelines, was performed for almost 20 min. Eventually, the patient recovered with sinus tachycardia and hypotension.

OUTCOME AND FOLLOW-UP

A right internal jugular vein central line was inserted and intravenous norepinephrine, epinephrine and phenylephrine infusions were used, in addition to fluid resuscitation (figure 3). Despite aggressive vasopressor therapy, the patient continued to be unstable with hypotension and severe lactic acidosis. An urgent echocardiography was performed, which showed a pulmonary artery pressure of 65 mm Hg and massive pericardial effusion with tamponade (figure 4). A pericardiocentesis was performed but it failed to improve the patient's haemodynamic state. Further, a cardiac arrest occurred. CPR failed to revive the patient, who was pronounced dead.

DISCUSSION

MMS is defined as acute right ventricular failure secondary to vascular compression after positive pressure ventilation.^{1 2} It was described in the 1970s by Dr Bittar who found a high-risk

Figure 3 Postintubation chest X-ray showing the endotracheal tube, right jugular central venous line, feeding tube, thoracostomy tube in place. Extensive bilateral airspace disease.

Figure 4 Subcostal view of transthoracic echo showing massive pericardial effusion with diastolic collapse of the right atrium and right ventricle.

of perioperative morbidity and mortality in association with general anaesthesia and the airway management of patients with anterior mediastinal mass.³

Cardiovascular and respiratory complications on account of the anterior mediastinal mass occur during induction of general anaesthesia. Supine position and loss of spontaneous breathing while starting the patient on positive pressure ventilation play a major role in the mechanism of these complications and subsequent hypoxia.³

Supine positioning of the patient with anterior mediastinal mass for intubation will cause an increase in critical airway pressure. This is explained by the reduction of the transverse thoracic diameter and cephalic displacement of the diaphragm. Furthermore, central blood volume will increase in the mediastinal mass in the supine position, thereby augmenting the size of the vascularised mediastinal tumour.⁴

Anaesthesia induction in patients with anterior mediastinal mass will shrink the transverse diameter of the thorax owing to a decrease in the inspiratory muscle tone, cephalic displacement of the abdominal contents secondary to loss of the abdominal muscle tone and relaxation of bronchial smooth muscles, leading to a major airway collapse. In addition, the craniocaudal movement of the diaphragm will be lost, or significantly diminished with the administration of neuromuscular blockers, as it happened in this case.⁴

Negative intrathoracic pressure caused by spontaneous breathing increases the venous return to the right ventricle, given the increment of the venous pressure gradient between the intrathoracic and extrathoracic vessels. With no such augmentation of the venous return, the cardiac output is compromised, which leads to hypotension.⁴

Extrinsic compression of both the pulmonary arteries and the superior vena cava will cause a decrease in the preload and an increase in the afterload.⁴ Moreover, owing to the supine positioning and anaesthetic induction, a reduction in the pulmonary artery diameter will impair pulmonary perfusion, resulting in severe hypoxaemia. Cardiac arrest will occur on account of acute right ventricular failure, as a result of increase of the pulmonary vascular resistance.⁴

Clinical examination of the patient might provide serious predictive signs of MSS like breathlessness, cough or syncope, especially when moving the patient from a vertical position to a horizontal position, or oedema of the upper trunk, and dilation of the neck veins indicating superior vena cava obstruction.⁵

Predictors of perioperative complications concerning respiratory or circulatory collapse in patients with a mediastinal mass are CT findings of $\geq 50\%$ tracheal compression or $\geq 130 \text{ cm}^3$ mass volume, presence of pleural effusion or pericardial effusion, presence of superior vena cava compression, pulmonary artery compression on one side, and atelectasis on the other side peak expiratory flow rate $\leq 40\%$ of the predicted value, and an obstructive or mixed pattern of pulmonary function test.^{1 6}

If general anaesthesia is inevitable, specific recommendations must be followed to avoid a life-threatening outcome during intubation⁴:

- ▶ Assess the risk and degree of severity through a detailed history and physical examination remains crucial. It is imperative to explore the best position for less dyspnoea and greater comfort prior to initiating general anaesthesia.
- ▶ Conduct a preoperative chest X-ray, CT and echocardiogram to ascertain details of mediastinal mass and nearby compressed structures.
- ▶ Classify risk of developing mediastinal mass syndrome.
- ▶ Intubation location should be in the operating room with preoperative consultation by a multidisciplinary team involved in specialised anaesthetics, intensivists and cardiothoracic surgeons.
- ▶ Make a cardiopulmonary bypass or extracorporeal membrane oxygenator and perfusionist available, as the patient may need rescue ventilatory support. Femoral venous cannulation should be ready prior to anaesthesia induction in high-risk patients.
- ▶ Maintain Fowler's position (sitting straight up or leaning slightly back and straight or bent legs) during induction of sedation or transportation through anaesthetic.
- ▶ Awake intubation with assistance of fiberoptic bronchoscopy is preferred.
- ▶ Avoid deep sedation and proceed with stepwise induction. Inhalational anaesthesia might be an alternative.
- ▶ Avoid muscle relaxants.
- ▶ Maintain spontaneous, low-tidal volume ventilation during intubation and after connecting to mechanical ventilation. Insert arterial line for haemodynamics monitoring.
- ▶ Initiate proper vascular access for patient in the groin. A large bore vein catheter should be used for fluid administration, prior to induction. A central venous catheter should preferably be inserted in the femoral area with the possibility of upper thoracic vein compression. It is preferable to have the femoral venous and arterial line on the same side, so as to save the other side for possible cardiopulmonary bypass.
- ▶ Continuously assess the effects of anaesthesia on respiratory and haemodynamic parameters.
- ▶ Maintain a good preload in case of hypotension or blood loss with volume replacement.
- ▶ Make available different sizes of endotracheal tubes and a flexible and rigid bronchoscope.

The ICU team realised that a simultaneous arterial and venous blood gas analysis could have been undertaken to evaluate the effect of cardiac tamponade on the patient's haemodynamics. A progressive increase in the difference in partial pressure of CO_2 between arterial (PaCO_2) and partial pressure of CO_2 venous (PvCO_2) blood usually precedes the haemodynamic compromise caused by cardiac tamponade. An increasing gap between PaCO_2 and PvCO_2 warrants an immediate drain of the pericardial effusion in the right clinical setting.

This may also have been used to differentiate between MMS and cardiac tamponade. Unfortunately, no blood gases were checked at this point, owing to the rapid deterioration in the patient's condition.

However, an urgent echocardiography was planned, and pericardiocentesis was eventually performed with no significant improvement in the patient's clinical condition.

Learning points

- ▶ Mediastinal mass syndrome (MMS) is a rare complication of anaesthesia induction for airway management, with a high-mortality rate.
- ▶ Intubation of patients with mediastinal mass must be approached preoperatively through appropriate evaluation by a multidisciplinary team.
- ▶ Airway management of patients with mediastinal mass should be carried out in the operation room. Awake fiberoptic endobronchial intubation is more favourable in induction by sedation.
- ▶ A rigid bronchoscope and cardiopulmonary bypass should be available as a rescue for patients who will undergo respiratory compromise.

Contributors IA-S managed the patient in the ICU and wrote the discussion and references. MS provided all the required images for the manuscript. MA-S performed Echo for the patient and added comments on the echo findings to the report. SA wrote the background, summary, case presentation and reviewed the manuscript.

Competing interests None.

Patient consent Obtained.

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